Venous thromboembolism in trauma patients: Standardized risk factors

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Purpose: This study was done to evaluate the use of published standardized risk factors for venous thromboembolism (VTE) in patients admitted to a trauma intensive care unit (ICU) and to derive guidelines for the use of low molecular weight heparin (LMWH) and surveillance venous Doppler ultrasound scanning (VDUS).

Methods: Patients were admitted to a regional trauma center ICU. Two periods were studied. Period 1 was a retrospective analysis of documented cases of VTE in the trauma registry from 1993 to 1995 (n = 39). The period was also a review of all patients admitted to a trauma ICU in 1994 without VTE who met the following criteria: age greater than 11 years, ICU stay of more than 36 hours, and survival of more than 72 hours (n = 227). Period 2 was a concurrent analysis of 1996 documented cases of VTE and similarly selected ICU admissions (VTE, n = 10; no VTE, n = 224). Risk factor scores (R1, admitting; R2, total) were calculated from the International Society for Cardiovascular Surgery/Society for Vascular Surgery reporting standards. The scores were cumulative by category and over time. The suitability of such standards was determined in period 1. The resulting therapeutic and surveillance guidelines were evaluated in period 2.

Results: Period 1 risk factor scores, R1 and R2, were correlated with the occurrence of VTE from χ^2 test ($P < .05$ and $P < .01$, respectively). Risk categories were grouped as low, moderate, and high. VTE was not observed in the low-risk group (0 to 2). Among all VTE (n = 49), 11 cases occurred in patients with moderate-risk scores and 38 in patients with high-risk scores. In 1994 and 1996, the selected groups were analyzed and the incidence rate of VTE was 4.7% in both years for the moderate-risk group and 2.5% and 4.8% for the high-risk group, respectively.

Most VTE cases (78%) received some form of prophylaxis (PRx), and 26% of cases had multiple methods of prophylaxis (MPRx). This included 80% of the cases that received unfractionated heparin.

In period 2, no pulmonary emboli (PE) occurred, in contrast to period 1, in which 16 of 39 cases of VTE (41%) were first seen with PE. In period 2, no patient receiving MPRx, including compression and LMWH, had VTE develop. Surveillance VDUS discovered 60% of 1996 cases in period 2. No PE were seen in period 2.

Conclusion: Standard risk factors were easily applied to the trauma patient at the bedside. Patients at low risk needed no PRx. Patients at high risk did best with both compression devices and LMWH. VDUS was recommended selectively in patients at high risk in whom multiple-method PRx could not be achieved. Patients at moderate risk required further study to define optimal PRx and need for surveillance VDUS. Intracaval devices were used prophylactically only twice. (J Vasc Surg 1998;28:250-9.)

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Although the trauma setting virtually defines major risk for venous thromboembolism (VTE), scanty data exist among prospective studies[1-9] to guide the surgeon in the selection of the optimal methods of prophylaxis (PRx) or in the use of surveillance diagnostic studies such as venous Doppler ultrasound scanning (VDUS).[1,4,10-14] Anticoagulation therapy with low molecular weight heparin (LMWH) appears to be evolving as

Table I. Occurrence of venous thromboembolism from 1993 to 1996 by low-risk, moderate-risk, and high-risk categories

All VTE	*1993 (No. PE)*	*1994 (No. PE)*	*1995 (No. PE)*	*1996 (No. PE)*	*Total (No. PE)*
Low risk 0-2	0	0	0	0	0
Moderate risk 3-5	2 (2)	4 (1)	1 (1)	4 (0)	11 (4)
High risk ≥6	19 (8)	3	10 (3)	6 (0)	38 (11)
Total	21	7	11	10	49 (15)
*Percent of all ICU admissions	4.5%	1.6%	2.2%	2.1%	

VTE, venous thromboembolism; *PE*, pulmonary emboli; *ICU*, intensive care unit. *Relative incidence rate of venous thromboembolism for all admissions is indicated on the bottom line.

the most effective chemical method.[1,2,8,15] This method is also the most expensive and may be limited in applicability, especially in cases of neurologic or major pelvic trauma. Compression devices are reported to have varying success in the trauma setting, particularly for neurologic trauma.[1,4-6,8,11,12,16,17] Data regarding multiple-method PRx are conflicting.[4,6,8]

The diagnosis of VTE is complicated by the frequent absence of symptoms in the continuum of its major manifestations.[18,19] VDUS, phlebography, and magnetic resonance angiography—all have been studied and advocated.[1-4,20-24] However, the overriding concern among these choices has been their cost and effectiveness.[1,2,8,10,11,13,25,26]

The lack of standardized risk factors and patient stratification according to risk does not allow comparisons between reports from different institutions. Although many studies accept the major risk factors defined by the National Institutes of Health and European consensus studies,[27,28] most define individual factors rather than a cumulative risk. As a result, data reduction tends to eliminate known risk factors, as seen in the recent summary of studies by Knudson.[4] Published reporting standards of risk, which are cumulative in their score,[29] have not been systematically applied. This study was begun to evaluate the use of the International Society for Cardiovascular Surgery / Society for Vascular Surgery (ISCVS / SVS)[29] reporting standards of VTE risk in the trauma setting. Guidelines then were derived for the use of single-method and multiple-method PRx and the indication for surveillance VDUS.

MATERIAL AND METHODS

All patients in this study were admitted to a level II trauma center serving northern Nevada and the eastern Sierra slope of California. Approximately 2000 patients were admitted for trauma annually during the study (92% blunt trauma, 8% penetrating). Intensive care unit (ICU) admissions for trauma varied from 430 to 470 per year; the general demographics were similar from year to year. A panel of 10 trauma surgeons staffed this unit, with 2 specialized trauma nurse coordinators.

In period 1, all documented cases of VTE were reviewed retrospectively from 1993 through 1995 (n = 39). In addition, patients in trauma ICU (age more than 11 years, survival more than 72 hours, and ICU stay more than 36 hours) who did not have VTE were studied retrospectively for 1994 as a denominator (n = 227). In period 2 for 1996, all cases of documented VTE (n = 10) and similarly selected ICU cases without VTE (n = 224) then were tracked concurrently.

Risk factors used were adopted from the reporting standards recommended by the ISCVS / SVS in 1988,[29] with slight modifications to suit the trauma setting. The risk factor grades were cumulative both at admission and over time as published. Delayed diagnoses and multiple operations (up to 3) were included in the total score, R2. The following factors and grades were defined:

Age: 0 to 39 years = 0, 40 to 69 years = 1, ≥70 years = 2

Long bone fracture: none = 0, soft tissue = 1, tibia, fibula, arm = 2, femur = 3, pelvis = 4

Immobilization: none = 0, 1 to 3 days = 1, >3 days = 2, paraplegia or quadriplegia = 3

Anesthesia time: none = 0, local or <45 minutes = 1, <3 hours = 2, >3 hours = 3

Prior VTE: none = 0, suspected = 1, proven = 2, multiple = 3

Individual risk factors that were not immediately apparent at admission or from the records, such as obesity or the presence and degree of heart disease or cancer, were simplified as present = 1 and absent = 0. The remaining recommended risk factors rarely were encountered and, therefore, were omitted from this analysis.[29]

Table II. Annual occurrence of venous thromboembolism in patients at moderate risk and high risk as related to prophylactic methods

Risk	*PRx*	*1993*	*1994*	*1995*	*1996*	*Total*
Moderate	None	1	2			3
Moderate	SCD	1	2	1	4	8
Moderate	MPRx	0	0	0	0	
High ≥6	None	3	0	0	0	3
High ≥6	SCD	13	2	6	4	25
High ≥6	MPRx	3	1	4 (2*)	2	10 (2*)
Total		21	7	11	10	49

PRx, prophylaxis; *SCD*, sequential compression device or air velocity index boot; *MPRx*, multiple-method prophylaxis (SCD and anticoagulants). *Number of patients who received low molecular weight heparin.

An initial score R1 was derived. This score was the sum of the various factors known to the trauma team on morning rounds after the patient was admitted to the ICU. In period 2, the PRx requirements and need for surveillance then were determined. Total risk R2 was the sum of all risk factors at the time of discharge, death, or a VTE event. From analysis of the descriptive scores in period 1 data, the risk categories were defined arbitrarily as follows: low = 0 to 2 factors, moderate = 3 to 5 factors, and high = 6 or more factors. A score of 15 was the maximum score possible. Direct venous injury and upper-extremity VTE were omitted to focus the analysis on lower-extremity VTE.

The staff agreed on guidelines for PRx as a result of period 1 descriptive data. These guidelines were accepted as follows: scores of 0 to 2 did not need PRx other than early ambulation and moderate-risk scores of 3 to 5 received compression devices, sequential compression devices or AVI foot compressors, which were placed in the operating room or ICU. Enoxaparin alone was considered an acceptable alternative. Compression devices and LMWH or Enoxaparin were advised for patients with risk scores of 6 or more. LMWH therapy was begun as soon as possible after admission, with 30 mg being given subcutaneously every 12 hours. Period 1 experience revealed that 8 of 10 patients for multiple-method PRx with documented VTE had received unfractionated heparin. In 1995, only 2 cases of VTE were documented with LMWH as the second method, which suggests more liberal use of this agent.

Whenever optimal PRx was not possible, routine VDUS was advised. Studies were done at days 5 to 7 and then weekly until patients were ambulant. VDUS was done in a registered noninvasive laboratory and interpreted independently by a panel of 9 vascular surgeons. Three of these participated on the trauma team, but they did not know which patients received PRx.

Surveillance lung scanning was not done. Intracaval devices were considered in patients at high risk with a score of 10 or more and, particularly, in patients with head injuries with a contraindication to anticoagulants and patients with major pelvic fractures. Otherwise, standard indications for deep venous thrombosis (DVT) or pulmonary emboli (PE) were used for filter placement.

Demographics for all trauma ICU admissions and the defined-risk ICU admissions were analyzed with Student's *t* test. Nonparametric data were compared with χ^2 test.

RESULTS

In period 1, the initial risk factor score, R1 ($P < .05$), and total risk factor score, R2 ($P < .01$), correlated with the occurrence of VTE by χ^2 test. The demographics of all trauma ICU admissions for the 4 years studied were similar except for the length of stay. Because of deliberate efforts of the trauma team and utilization committees, the length of stay was reduced by approximately 24 hours in 1996.

Table I shows the occurrence of VTE as classified by year and risk categories. To date, no instance of VTE has been detected in the low-risk group (0 to 2 factors). Eleven cases of VTE were seen in the moderate-risk group; 4 had PE in period 1, and 1 case was fatal. Most VTE cases occurred in the high-risk group (n = 38, 77.5%). Twelve PE were seen in period 1, and 4 cases were fatal.

In period 1, VTE cases were documented by means of VDUS or ventilation-perfusion scanning or autopsy in the 5 cases of fatal PE. For the years with a selected baseline group, the incidence rate of VTE was 4.7% in the moderate-risk group for both 1994 and 1996. The incidence rate for the high-risk

group was 2.5% and 4.8%, respectively. The infrequency of surveillance VDUS in period 1 prohibits quantitative conclusions regarding the true incidence rate of VTE in that time. However, once again, PE occurred in 5 instances as the first indication of VTE in period 1. No cases of PE were documented in 1996. The occurrence of VTE by year is shown for year to year comparison, with all unselected ICU admissions (range, 430 to 470) for 1993 (n = 21, 4.5%), 1994 (n = 7, 1.6%), 1995 (n = 11, 2.2%), and 1996 (n = 10, 2.1%). Essential population demographics were age, Injury Severity Scores (ISS), Glascow Coma Scale (GCS), and ICU stay. These were similar, except as noted for length of stay in 1996.

Table II shows the annual occurrence of VTE cases by risk category and PRx. No cases of VTE were observed in the low-risk group. Six cases of VTE received no PRx (period 1); these cases were distributed equally between moderate-risk and high-risk scores. Eight of the 11 moderate-risk VTE cases had used compression devices. Two thirds of the high-risk group with VTE received compression devices only. In period 1, 10 cases of VTE received multiple-method therapy, but in only 2 instances in 1995 was the second method LMWH. The remainder received unfractionated heparin at 8-hour or 12-hour intervals.

Table III shows selected designated-risk ICU cases without VTE admitted in 1994 and 1996 on the basis of the criteria of age (>11 years), ICU stay (>36 hours), and survival (>72 hours). This analysis was done to select the patients with the highest risks. The mode of PRx is illustrated along with the risk category. The number of patients at high risk who received only compression devices decreased from 68 (59%) in period 1 to 44 (37.3%) in 1996. At the same time, the number of patients who received multiple-method PRx increased from 43 (37%) to 71 (60.2%; $P < .01$, by χ^2 test). Enoxaparin was used as the additional method in 58% of cases in 1994 and 94.4% of cases in 1996 ($P < .01$, by χ^2 test).

Table IV indicates the percentage VTE by PRx and risk category for the 2 years with baseline denominator. The numbers do not permit statistical analysis. The relatively even distribution of VTE between patients at moderate and high risk in the even years, compared with the greater proportion of patients at high risk with VTE in the odd years, is noted. No documented cases of VTE were seen in the low-risk group or in those patients culled from the selected ICU denominator group.

The selected ICU admissions (1994 vs 1996) without VTE were essentially similar in average age (37 vs 36.8 years), ISS (19 vs 19.1), and ICU length of stay (7.9 vs 7.0 days). Overall, the patients for VTE were older, by 42 years, and had longer ICU stays, by 17.3 days. No significant differences in ISS were found in patients with and without VTE (average with VTE, 18; without VTE, 19). Long ICU stays are reflected in the score, particularly in immobilization. The numerically based guidelines for PRx derived from period 1 data were presented and discussed by the trauma panel in late 1995. Emphasis then was placed on the use of LMWH rather than unfractionated heparin. At the same time, the use of routine surveillance VDUS was recommended whenever optimal PRx was not or could not be achieved; nearly all patients received compression devices. In period 2, compliance with guidelines occurred in 89% of the cases. The noncompliant cases (26 of 234 admissions) included 2 patients in period 2 in whom VTE occurred. Noncompliance was tabulated when no anticoagulation therapy was used, and the VDUS surveillance guidelines were not observed. Bleeding and high-risk neurologic injury were the most common reason for no Enoxaparin.

Surveillance VDUS increased in period 2 and was performed in 72 instances in 1996 as compared with 44 times in 1994. In 1993, only 1 of the 21 VTE was discovered with surveillance as compared with 60% of those discovered in 1996. In those 4 years, 10 patients had DVT confined to the calf, 60% of which were discovered with surveillance. Thirty patients exhibited DVT in the proximal vessels, with or without calf involvement. Twenty-four patients had DVT that was diagnosed clinically. Only 6 cases were discovered with surveillance, whereas 50% of cases of proximal DVT in 1996 were discovered with surveillance. The location of DVT was unknown in 9 cases of PE in which the VDUS study was either not done or peripheral extremity thrombus was not shown.

Vena cava filters were used infrequently in this experience. Of the 14 placed, only 2 were done prophylactically. Twelve were inserted for standard indications, such as DVT or PE when anticoagulation therapy was contraindicated. Complications after filter placement included the development of 3 new instances of DVT, 1 extension of an established DVT, and 1 nonfatal PE.

DISCUSSION

The aim of this study was to simplify and quantify risk factors to allow the rational selection of

Table III. Prophylaxis used in selected cases* without venous thromboembolism during 1994 and 1996 in low-risk, moderate-risk, and high-risk categories

1994	*No PRx*	*SCD*	*MPRx*	*LMWH*	*Total*
Low risk 0-2	9	22	0	0	31
Moderate risk 3-5	13	58	10	2	81
High risk ≥6	4	68	43**	25**	115
Total	26	148	53		227
1996	*No PRx*	*SCD*	*MPRx*		*Total*
Low risk 0-2	12	12	0	0	24
Moderate risk 3-5	7	68	7	4	82
High risk ≥6	3	44	71**	67**	118
Total	22	124	78		224

PRx, prophylaxis; *SCD,* sequential compression device or air velocity index boot; *MPRx,* multiple-method prophylaxis (SCD and anticoagulants); *LMWH,* low molecular weight heparin.

*In these selected cases, age was >11 years, intensive care unit stay was >36 hours, and survival was >72 hours.

** Note increased prevalence of low molecular weight heparin use and multiple-method prophylaxis for high-risk group. $P < .01$ by χ^2 test for each.

methods of PRx for VTE and selective use of VDUS in VTE detection. This study did not address desirability of one particular form of PRx over another. The relatively low incidence rate of VTE overall reflects the staff education[30] and use of conferences devoted to VTE PRx by the trauma service. The first conference preceded data collection begun in 1993, with yearly updates of the data. As a result, the applicability of the standardized risk factors among this group was familiar and more readily accepted.

The true incidence rate of VTE is obscured, particularly in period 1 where data collection was retrospective, surveillance VDUS was sporadic, and overall denominators were unavailable for analysis for 1993 or 1995. The apparent decrease in VTE severity in period 2 only suggests the efficacy of MPRx. Surveillance-diagnosed silent VTE clearly increased (60% in 1996).

A close relationship between trauma and VTE initially was described by McCartney[31] as early as 1934. Successful prevention of VTE, with heparin in surgical cases, was summarized by Murray[32] in 1947. An unacceptably high incidence rate of VTE in trauma patients without PRx has been documented by Sevitt and Gallagher[33] and Kudsk et al.[24] Most recently, Geerts et al[2] showed an incidence rate of up to 77% of DVT when major head injury and lower-extremity fracture were combined. Geerts concluded that all trauma victims with an ISS of more than 9 should receive some form of PRx, confirming the recommendation by Hoyt and Swegle[34] that all patients in ICU should have VTE PRx.

Discrepancies in the literature derive from a lack of a standardized definition of high risk in the trauma patient. Trauma enhances risk for VTE. Lack of quantification renders comparisons of the results reported by different institutions difficult. The high costs of the emerging optimal PRx, such as LMWH,[35,36] and of surveillance VDUS[10,11,20] make rational selection an important consideration. The current system, graded by severity, cumulative by risk factors, and on the basis of published reporting standards (ISCVS/SVS Subcommittee on Reporting Standards in Venous Disease),[29] was found to be both practical and easily applied in the clinical setting.

A graded cumulative risk scoring system for VTE PRx is not original and was applied to general surgery by Farmer and Smithwick[37] in 1950. This system subsequently was adapted successfully for orthopedic trauma by Tubiana and Duparc[38] in 1961. Both authors cited cost and complexity of PRx as justifying the need for a selective process. The correlation with VTE of the current modification of the ISCVS/SVS scoring system in period 1 was clear. The concurrent application of scores to each new ICU admission in period 2 proved to be easily accomplished, and different team members readily agreed on scores.

Subsequent updating of the score allowed the inclusion of additional data as the clinical course evolved. The second risk, R2, assessed cumulative risk for each surgical procedure when multiple operations were required. About 20% of patients were moved into a higher risk category by a delayed or second operative procedure, such as spine fixation. The failure to react to the upgraded score possibly resulted in the occurrence of VTE in 2 of the period 2 cases. No scoring provision, however, exists for a

Table IV. Incidence rates of venous thromboembolism in years with baseline data

Year	*1994*				*1996*			
	VTE/no. patients		*VTE/no. patients*		*VTE/no. patients*		*VTE/no. patients*	
Risk	*SCD*	*%*	*MPRx*	*%*	*SCD*	*%*	*MPRx*	*%*
Moderate	2/60	3.3	0/10		4/72	5.5	0/7	
High	2/70	2.9	1*/44	2.3	4/48	8.3	2*/73	2.8

VTE, venous thromboembolism; *SCD*, sequential compression device or air velocity index boot; *MPRx*, multiple-method prophylaxis (SCD and anticoagulants). In 1994, 2 of 7 (13.3%) venous thromboembolism events were in patients at moderate risk with no prophylaxis. Venous thromboembolism was not documented in the patients at low risk or in the patients selected from the intensive care unit group.
*Standard heparin in all cases.

physiologic "second-hit", such as septic shock, and perhaps this refinement is needed. Such events can prolong immobility and trigger the clotting cascade. A second underassessment occurred in a young patient with spinal cord injury caused by a diving accident who did not undergo major surgery.

The basic risk groups, low, moderate, and high, were derived in period 1 by retrospective analysis of documented VTE for 3 years. The selection of the group of patients in the ICU from 1994 sought to eliminate young children, overnight ICU stays, and early mortality rates. Overall, the VTE occurred as follows: none in low-risk group, 25% in moderate-risk group, and 75% in the high-risk group.

Discrimination by score between VTE occurrence and no VTE was not observed in the preliminary data from the multi-institutional study by Greenfield et al.[8] The complexity of the scoring procedure suggested in that study may make application at the bedside by trauma personnel more difficult. Because we made no preliminary determination of heparin/no heparin, we cannot compare our data with several other reports that address this issue.[1,3,8] Most of our patients at high risk who received compression therapy alone would fall into the no-heparin category, according to the criteria used by Knudson[1] and Greenfield,[8] but not necessarily that used by Geerts.[3]

The surveillance program in period 2 appears to have contributed to the absence of PE and is consistent with the recent report by Brasel et al.[13] These authors suggest that VDUS screening was more cost-effective than routine caval filter placement. The trend for more of the patients at high risk in 1996 to receive multiple-method PRx, particularly the use of LMWH (Enoxaparin), was statiscally significant. Enoxaparin was chosen because of the strong support from the orthopedic literature combined with experiences with heparin failure as the second method early in the study. This choice is supported by the Canadian study by Geerts,[3] which shows the superiority of LMWH over unfractionated heparin. Knudson et al[1] also have shown that Enoxaparin was most effective in their patients who were candidates for anticoagulants. The preliminary data of the Greenfield et al[8] effort also seem to confirm this opinion.

The major problem in these studies of trauma and VTE is the determination of eligibility for use of anticoagulants in patients with neurotrauma. The Canadian[3] study included "…all but frank intracranial bleeding on computed tomographic scanning…or bleeding that remained uncontrolled 36 hours after the injury" or "indications of coagulopathy". The study included patients with "cerebral contusion, localized petechial hemorrhages, or diffuse axonal damage. "Knudson[1] criteria excluded a GCS of less than 8, spinal cord injury, and nonoperative management of liver and spleen injuries. Greenfield[8] excluded intracranial bleeding by means of computed tomography, incomplete or progressing spinal cord injury, pelvic trauma requiring 2 or more units of blood, nonoperative management of liver and spleen injuries, and other considerations.

Hamilton et al[39] note that "…adequate prophylaxis is underutilized in contemporary neurosurgical practice" and that LMWH may be used safely. Further dialogue with our neurosurgical colleagues might increase the routine use of multiple-method PRx in this problematic group of patients. For the high-risk head-injury group with a contraindication to LMWH, a decision rests between compression devices and surveillance versus intracaval filters, as discussed by Rodgers,[26] Winchell,[25] Kansarina,[40] and Patton.[41] Knudson[1,4] found in her studies that sequential compression devices were helpful in head injuries as compared with no PRx. Gersin[9] concluded that such devices were "not entirely effective". We concur with the latter opinion inasmuch as 25 of 38 patients at high risk who were documented in the current study with VTE had this form of PRx

only. More extensive use of LMWH might solve part of this problem provided that costs are not excessive. The multi-institutional study of Greenfield et al[8] will further clarify the use of the filter in these situations.

A troublesome finding in the current study was the relatively high frequency of VTE in the moderate-risk group in whom compression devices would have been thought to be adequate. These occurrences were clustered in 1994 and 1996. Because the basic demographics for all of the trauma ICU admissions in the 4 years were not significantly different, this variance is not explained. VTE in the moderate-risk group accounted for 4 PE, 1 case of which was fatal. With the favorable experience in the high-risk categories with the use of LMWH, increased use of LMWH in the moderate-risk group is suggested. With an increased use of LMWH, increased cost requires consideration when used in addition to sequential compression. Certain factors might offset drug cost. As confidence and use of LMWH increases, the need for surveillance VDUS might decrease. Also, early conversion of patients to warfarin PRx can be considered as soon as a reliable route of enteral feeding is established.

Most of our cases of VTE involved thrombi in the proximal veins, and only 10 of 30 documented DVT cases were isolated to the calf. Of these, 60% were discovered by means of VDUS. Once found, however, such calf thrombi require attention. Loher et al[42] have documented the tendency of crural thrombi to propagate. A number of studies have implicated infracrural thrombi as the sole source of PE.[43-45] Currently, we advise upgrading the method of PRx, or therapy, consonant with the clinical status when the calf vein thrombi are found. This would include addition of LMWH.

This observation of numerous calf thrombi in addition to proximal thrombi confirms the use of surveillance VDUS in trauma patients. Our selective use of VDUS is consistent with the recommendations of Meyers et al,[10] Napolitano et al,[11] and Knudson.[4] The frequency of "unsuspected" DVT in the current series, including 5 of 25 in proximal vessels, is comparable with other reports and further supports the wisdom of surveillance. The current findings do not support a purely clinical approach to VTE PRx as advocated by others.[46] Our limited use of intracaval filters reflects our concern for complications as documented by Patton.[41] We support Greenfield's opinion that DVT is the primary problem.[47] Cava filter placement seems to be the safest option when other methods are not applicable or contraindicated in high-risk situations.

The risk scoring system functions well on a day-to-day basis and can be applied easily by various observers. The overall occurrence of VTE after 1993 was relatively constant because the diagnosis of silent VTE was enhanced by increased use of surveillance VDUS in period 2. PE were absent in period 2, possibly related to the intensification of PRx and efforts at early diagnosis of DVT. We have been cautious in interpretation of this clinical study involving the patients who were typical and heterogeneous and variations in staff management in a trauma center. A randomized study that focuses on moderate-risk PRx appears indicated.

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DISCUSSION

Dr. Kenneth E. McIntyre Jr (Galveston, Tex.) The purpose of this study, as stated by the authors, was to evaluate the use of standardized risk factors for venous thromboembolism in trauma patients, to develop guidelines for the use of venous duplex ultrasound scanning for surveillance, and to study the use of single-method and multiple-method prophylaxis of venous thromboembolism. This study is relevant because pulmonary embolism has been observed in as many as 22% of patients for trauma and is the third most common cause of death in those patients who survive more than 24 hours. The challenge is formidable: to develop effective prophylaxis for venous thromboembolism in a patient population that may have a higher than expected risk of bleeding.

The subjects of this study were patients for trauma who were admitted to the intensive care unit of a level II trauma center in Northen Nevada. During period 1 from 1993 to 1995, a retrospective review of all documented cases of venous thromboembolism and a correlation with the presence of known risk factors were performed. The authors modified the International Society for Cardiovascular Surgery / Society for Vascular Surgery risk factors and did not include pregnancy or postpartum states, hormonal therapy, or prethrombotic states as additional risk factors. On the basis of these data, the authors devised 3 risk categories (low, moderate, high) and made recommendations for deep venous thrombosis prophylaxis to be evaluated prospectively during period 2, in 1996. Patients considered to be at low risk received no prophylaxis, those considered at moderate risk received leg compression devices, and those considered at high risk received both leg compression devices and low molecular weight heparin, or Enoxaparin. They compared the incidence rates of venous thromboembolism for every year in each risk category and recorded the prophylaxis measures used. All of the documented cases of venous thromboembolism occurred in the moderate-risk and high-risk groups.

There are a few problems with this study, and I will try to elucidate them for you. This group was not homogeneous because of the nature of the injuries and because 10 different surgeons were responsible for the care. All of the patients did not undergo routine duplex scanning at recommended times. It was only advised when prophylaxis was not possible, and it is unclear from the manuscript exactly what prompted ordering of the duplex scanning. Because all patients did not undergo routine duplex surveillance, the true incidence rate of venous thromboembolism as related to risk factors was unknown or at best underestimated. Only selected patients who were admitted to the intensive care unit for trauma were evaluated concurrently, and apparently, once patients left the hospital, they were not evaluated further. There was no standard protocol for prophylaxis either. Compliance with recommended guidelines occurred in only 85% of the cases. Because routine duplex scanning was not done, it was also difficult to evaluate the efficacy of prophylaxis. Vena caval filters were used so infrequently that evaluation of them as a tool for prophylaxis could not be done.

Are there positive attributes of this study? The risk stratification that was used seemed to be effective in predicting thromboembolism and seemed applicable for personnel treating the patient for trauma. The surveillance program, which was expanded during period 2, contributed to the diagnosis of silent proximal deep venous thrombosis and may have contributed to the reduction in pulmonary embolism. Thromboembolism prophylaxis may not have been as successful as predicted because of prolonged intervals between injury and initiation of prophylaxis.

I have several questions for the authors.

1. Was a single negative duplex scanning considered adequate for patients without thromboembolism prophylaxis, or were these patients scanned more than once during their hospitalization?
2. Were patients followed-up once they left the hospital?
3. If so, did any patients develop venous thromboembolism after they went home?
4. Why did 78% of patients in period 1 sustain venous thromboembolism despite some form of prophylaxis?
5. Why were sequential compression devices alone apparently unsuccessful in preventing venous thromboembolism?
6. How long after injury was thromboembolism prophylaxis begun, and when was it terminated?
7. What was the cost of prophylaxis when Enoxaparin was used?
8. What treatment are you advising for patients with tibial vein thrombosis and no documented extension into the popliteal vein?
9. How did you follow patients with casts, dressings, or open wounds on the legs?
10. What is your current recommendation for venous duplex surveillance in patients with trauma?

The authors have attempted to predict which patients with trauma are at risk for thromboembolism by using a standard risk factor analysis that seems to have some merit. However, because of a lack of following recommended protocols for routine duplex surveillance and standard prophylaxis, I am not convinced of the type of prophylaxis for venous thromboembolism that is preferred. Moreover, the strong recommendation of the authors for the use of Enoxaparin in patients with moderate risk cannot be justified on the basis of their data. I would encourage the authors to continue their work with protocols for duplex surveillance at standard times and routine prophylaxis measures for each risk category. In addition, patient follow-up after hospitalization is important to document the

natural history of patients for trauma with and without thromboembolism after intensive care unit admissions.

Dr. H. Treat Cafferata. Your first question asks whether a single negative duplex scanning is adequate. We thought is was not. The scannings were indicated in the patients usually because the patients had head injuries and would not tolerate Enoxaparin or because the neurosurgeons would not permit us to use Enoxaparin. Those patients were followed-up at least at the end of the 1st week and hopefully at 1-week to 2-week intervals. I do not think that there is any established protocol for how often venous Doppler ultrasound scanning should be done, and in our institution, it costs the patient approximately $500 for each scan. So, this whole study tried to design reasonable boundaries at least for doing these procedures and keeping within the cost controls that exist.

Our patients were followed-up until discharge. As you know, there is much open space in Nevada. Many of the accidents occur with tourists as single-car rollovers in the middle of nowhere. Follow-up after discharge is difficult as they leave our community and return home.

If no deep venous thrombosis was documented in the intensive care unit, the patients were followed-up on the floor by the trauma nurse coordinators. We did not have any incidences of which we are aware of people developing this at home. I understand there are a number of papers that advocate this.

The prophylaxis therapy was begun as soon as possible after injury. Sequential devices whenever possible were put into the operating rooms. The Enoxaparin was started at least within 36 hours if at all possible. Again, as I say, there were other factors that governed it.

As far as tibial veins are concerned, our treatment recommendations are pretty much on the basis of the conditions under which they are documented. So, if the patient was one of those in the low-risk or moderate-risk category that had not had any form of therapy, we would recommend at least sequentials or prophylactic heparin levels.

If they developed where the patient had sequentials or was on an anticoagulant, we probably would recommend the next step of therapy. There are documented issues in some people that these will embolize in the pulmonary system. Even Norman Browse says that they are nonfatal, but that small embolus could be fatal in a trauma patient on a ventilator with adult respiratory distress syndrome. So, we are a little aggressive about that.

Why do sequentials not work? Part of the reason is because they are not always used on the floor. Another part of the reason is that they are just not totally effective.

How do we deal with patients with bandages, casts, or external fixators? Well, we deal the best we can. We are just trying to treat the patient as we see them. So, we do not always have perfect data, but we cannot always get that.

Dr. Kaj H. Johansen (Seattle, Wash.). Kudos to Dr. Cafferata and colleagues for confronting an important issue that is currently being pursued primarily in the natural history realm.

It is with some alarm, therefore, that I hear what I believe is an uncritical assumption of the value of low molecular weight heparin as a prophylactic agent. I am aware of the data on which this is based, but I am unclear whether the statistical significance that suggests the benefit of low molecular weight heparin over unfractionated heparin is equalled by a clinical significance as well.

I am not at all sure that, at pennies rather than many hundreds of dollars, we should not be simply giving these patients unfractionated heparin if in-hospital prophylaxis is what we have in mind.

Dr. Cafferata. Thank you. You are right. Although I am not sure exactly, I was told that it cost our hospital approximately $17 a dose for Enoxaparin. This is, of course, well above what is cost effective in Canada. In fact, it is almost three times that. However, all of the data in the orthopedic literature and in a few current trauma studies suggest that the superiority does lie with low molecular weight heparin.

So, for the patients at high risk, I do not feel that there is a problem there. As soon as we get these people to where there is a reliable enteral route, we will start to switch to Coumadin.